13) Responses to vasoconstrictile agents in the aortas and mesenteric arteries of M-SHRSP, SHRSP and WKY, with special reference to aging. Tamaki Nakata, Kunio Abiko, Hiroshi Ikawa and Aritomo Suzuki. Department of Pharmacology, Kinki University School of Medicine, Osaka-fu 589.

It has been reported that M-SHRSP developed from SHRSP dies by stroke at the early stages. In this experiment, using the isolated blood vessels from M-SHRSP, we examined the contractions by drugs in comparison with those of SHRSP and WKY from the standpoint of aging. From the results, we have discussed whether the abnormal responses are due to the heredity or the secondary by high blood pressure.

Materials and Methods. M-SHRSP, SHRSP and WKY at 3, 6 and 12 weeks of age (W) were used. The isolated aortas and mesenteric arteries were hung in a Magnus' instrument containing Locke's solution maintained at 37°C. The change of tonus by stimulating agents was isometrically recorded on a recorder through a force-displacement transducer. The drugs used are as follows: noradrenaline (Nad), serotonin (5-HT), angiotensin II (Ang), prostaglandin F_{2α} (PGF), K⁺ and Ca²⁺.

Results in aorta. The contractions by Nad, Ang, PGF, 5-HT, K⁺ and Ca²⁺, in the aortas of M-SHRSP and SHRSP at 3 W were larger than those of WKY at the same W. Especially the contraction by Ca²⁺ were much enhanced. The similar tendency was found at 6 W. At 12 W the contractions by K⁺ and Ca²⁺ were enhanced in both M-SHRSP and SHRSP. The contractions by Nad, PGF and 5-HT were depressed in M-SHRSP, while these were still enhanced in SHRSP.

Results in mesenteric arteries. At 3 W contractions by drugs were larger in M-SHRSP than in SHRSP and WKY, showing no difference between SHRSP and WKY. At 6 W contractions by the drugs were enhanced in M-SHRSP and SHRSP, especially more remarkable in SHRSP. Although the contractions by K⁺ and Ca²⁺ were still enhanced in M-SHRSP at 12 W, the contractions by Nad, Ang, 5-HT and PGF were reduced. While, in SHRSP at the same W the contraction by Ang alone was reduced.

Discussion. Contractions by the drug in M-SHRSP were enhanced at the early stages. It has been reported that the blood pressures of M-SHRSP, SHRSP and WKY at 3 W are 150, 110 and 100 mmHg, respectively. Thence the increased contractions in M-SHRSP at 3 W might be to the secondary by the high blood pressure, because the thickening of media was already found. This view may be supported by the fact that the contractions of mesenteric artery of SHRSP were almost the same as those in WKY, and may coincide with the thought by Folkow. But this thought may be correct partly. The contractions in the aorta of SHRSP at 3 W were already larger compared with those in WKY. It is reported that the blood pressure at 6-7 W of SHRSP is almost 120 mmHg. Thus, this degree of blood pressure dose not seem to be high. The fact that the contractions in SHRSP at 6 W were more enhanced compared with those in WKY in both aorta and mesenteric artery might mean that the increased contraction might be hereditary.

The enhanced contraction found in immature M-SHRSP and SHRSP might be more developed at the early stages than the elevated blood pressure. In M-SHRSP the contractions by Nad, PGF and 5-HT were reduced at 12 W. It has been reported that the contractions by Ang and Nad were reduced in the aorta of SHRSP at 6 month old and that the reduction found in the aorta and mesenteric artery of M-SHRSP at 12 W might be due to the desensitization of receptors. On the other hand, there is also a thought that the muscle contractile element in M-SHRSP was disturbed, which is denied by the fact that the contractions by K⁺ and Ca²⁺ were not reduced.

Conclusion. Responses to the vasoconstrictile agents were enhanced at the early stages such as 3 W, and increased with aging. At 12 W the contractions by Nad, PGF, Ang and 5-HT were, however reduced, although the contractions by K⁺ and Ca²⁺ were still enhanced.